

WEST Search History

DATE: Monday, November 15, 2004

Hide?	Set Name	Query	Hit Count
		<i>DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L3	L2 and L1	78
<input type="checkbox"/>	L2	MKK6 or (map adj kinase adj kinase adj 6)	198
<input type="checkbox"/>	L1	antisense or anti-sense	56748

END OF SEARCH HISTORY

/H = Help

/L = Logoff

/NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).

? b 410

```
19oct04 15:05:43 User214465 Session D1448.1
      $0.00      0.198 DialUnits FileHomeBase
$0.00 Estimated cost FileHomeBase
$0.00 Estimated cost this search
$0.00 Estimated total session cost      0.198 DialUnits
```

File 410:Chronolog(R) 1981-2004/Sept
(c) 2004 The Dialog Corporation

Set	Items	Description
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---	-----	-----
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? set hi ;set hi

HIGHLIGHT set on as ''

HIGHLIGHT set on as ''

? b 5,155,357,399

```
19oct04 15:05:55 User214465 Session D1448.2
      $0.00      0.097 DialUnits File410
$0.00 Estimated cost File410..
$0.05 TELNET
$0.05 Estimated cost this search
$0.05 Estimated total session cost      0.295 DialUnits
```

SYSTEM:OS - DIALOG OneSearch

File 5:Biosis Previews(R) 1969-2004/Oct W2

(c) 2004 BIOSIS

File 155:MEDLINE(R) 1951-2004/Oct W3

(c) format only 2004 The Dialog Corp.

File 357:Derwent Biotech Res. _1982-2004/Oct W4

(c) 2004 Thomson Derwent & ISI

File 399:CA SEARCH(R) 1967-2004/UD=14117

(c) 2004 American Chemical Society

*File 399: Use is subject to the terms of your user/customer agreement.

Alert feature enhanced for multiple files, etc. See HELP ALERT.

Set	Items	Description
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? s map(w)kinase(w)kinase(w)6 or mkk6

161645 MAP

614069 KINASE

614069 KINASE

3526401 6

214 MAP(W) KINASE(W) KINASE(W) 6

557 MKK6

S1 666 MAP(W) KINASE(W) KINASE(W) 6 OR MKK6

? s s1 and (probe? or antisense?)

666 S1

426569 PROBE?

83041 ANTISENSE?

S2 20 S1 AND (PROBE? OR ANTISENSE?)

? rd s2

...completed examining records

S3 14 RD S2 (unique items)

? s s3 not py=2001:2004

14 S3

6803397 PY=2001 : PY=2004

S4 5 S3 NOT PY=2001:2004

? t s4/7/all

4/7/1 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

Connecting via Winsock to Dialog

Logging in to Dialog

Trying 31060000009998...Open

DIALOG INFORMATION SERVICES

PLEASE LOGON:

ENTER PASSWORD:

Welcome to DIALOG

Dialog level 04.17.00D

Last logoff: 14oct04 13:59:28

Logon file405 19oct04 15:05:42

*** ANNOUNCEMENT ***

--Connect Time joins DialUnits as pricing options on Dialog.
See HELP CONNECT for information.

--SourceOne patents are now delivered to your email inbox
as PDF replacing TIFF delivery. See HELP SOURCE1 for more
information.

--Important Notice to Freelance Authors--
See HELP FREELANCE for more information

NEW FILES RELEASED

***Beilstein Abstracts (File 393)

***Beilstein Facts (File 390)

***Beilstein Reactions (File 391)

***F-D-C Gold/Silver Sheet (File 184)

***BIOSIS Toxicology (File 157)

***IPA Toxicology (File 153)

UPDATING RESUMED

RELOADED

***Toxfile (File 156)

REMOVED

***Textile Technology Digest (File 119)

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
>>> of new databases, price changes, etc. <<<

* * *

SYSTEM:HOME

Cost is in DialUnits

Menu System II: D2 version 1.7.9 term=ASCII

*** DIALOG HOMEBASE(SM) Main Menu ***

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

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0012679644 BIOSIS NO.: 200000397957

Antisense inhibition of MAP kinase kinase 6
expression

AUTHOR: Monia Brett P; Cowsert Lex M

JOURNAL: Official Gazette of the United States Patent and Trademark Office

Patents 1232 (1): Mar. 7, 2000 2000

MEDIUM: e-file

ISSN: 0098-1133

DOCUMENT TYPE: Patent

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Antisense compounds, compositions and methods are provided for modulating the expression of **MAP kinase kinase** *****6*****. The compositions comprise *****antisense***** compounds, particularly **antisense** oligonucleotides, targeted to nucleic acids encoding *****MAP***** *****kinase***** *****kinase***** *****6*****. Methods of using these compounds for modulation of **MAP kinase kinase** 6 expression and for treatment of diseases associated with expression of *****MAP***** *****kinase***** *****kinase***** *****6***** are provided.

4/7/2 (Item 2 from file: 5)

DIALOG(R)File 5: Biosis Previews(R)

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0011649415 BIOSIS NO.: 199800443662

Activation of p38 MAP kinase and JNK but not ERK is required for erythropoietin-induced erythroid differentiation

AUTHOR: Nagata Yuka; Takahashi Noriko; Davis Roger J; Todokoro Kazuo
(Reprint)

AUTHOR ADDRESS: Tsukuba Life Sci. Cent., Inst. Physical Chem. Res., 3-1,
Koyadai, Tsukuba, Ibaraki 305-0074, Japan**Japan

JOURNAL: Blood 92 (6): p1859-1869 Sept. 15, 1998 1998

MEDIUM: print

ISSN: 0006-4971

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: p38 MAP kinase (p38) and JNK have been described as playing a critical role in the response to a variety of environmental stresses and proinflammatory cytokines. It was recently reported that hematopoietic cytokines activate not only classical MAP kinases (ERK), but also p38 and JNK. However, the physiological function of these kinases in hematopoiesis remains obscure. We found that all MAP kinases examined, ERK1, ERK2, p38, JNK1, and JNK2, were rapidly and transiently activated by erythropoietin (Epo) stimulation in SKT6 cells, which can be induced to differentiate into hemoglobinized cells in response to Epo. Furthermore, p38-specific inhibitor S8203580 but not MEK-specific inhibitor PD98059 significantly suppressed Epo-induced differentiation and **antisense** oligonucleotides of p38, JNK1, and JNK2, but neither ERK1 nor ERK2 clearly inhibited Epo-induced hemoglobinization. However, in Epo-dependent FD-EPO cells, inhibition of either ERKs, p38, or JNKs suppressed cell growth. Furthermore, forced expression of a gain-of-function **MKK6** mutant, which specifically activated p38, induced hemoglobinization of SKT6 cells without Epo. These results indicate that activation of p38 and JNKs but not of ERKs is required for Epo-induced erythroid differentiation of SKT6 cells, whereas all of these kinases are involved in Epo-induced mitogenesis of FD-EPO cells.

4/7/3 (Item 3 from file: 5)

DIALOG(R)File 5: Biosis Previews(R)

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0011610155 BIOSIS NO.: 199800404402

p38 Mitogen-activated protein kinase mediates the transcriptional induction of the atrial natriuretic factor gene through a serum response element: A potential role for the transcription factor ATF6

AUTHOR: Thuerlauf Donna J; Arnold Nichole D; Zechner Dietmar; Hanford Deanna S; Demartin Kelli M; McDonough Patrick M; Prywes Ron; Glembotski Christopher C (Reprint)

AUTHOR ADDRESS: Dep. Biol., San Diego State Univ., San Diego, CA 92182, USA
**USA

JOURNAL: Journal of Biological Chemistry 273 (32): p20636-20643 Aug. 7, 1998 1998

MEDIUM: print

ISSN: 0021-9258

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: In various cell types certain stresses can stimulate p38 mitogen-activated protein kinase (p38 MAPK), leading to the transcriptional activation of genes that contribute to appropriate compensatory responses. In this report the mechanism of p38-activated transcription was studied in cardiac myocytes where this MAPK is a key regulator of the cell growth and the cardiac-specific gene induction that occurs in response to potentially stressful stimuli. In the cardiac atrial natriuretic factor (ANF) gene, a promoter-proximal serum response element (SRE), which binds serum response factor (SRF), was shown to be critical for ANF induction in primary cardiac myocytes transfected with the selective p38 MAPK activator, ***MKK6*** (Glu). This ANF SRE does not possess sequences typically required for the binding of the Ets-related ternary complex factors (TCFs), such as Elk-1, indicating that p38-mediated induction through this element may take place independently of such TCFs. Although p38 did not phosphorylate SRF in vitro, it efficiently phosphorylated ATF6, a newly discovered SRF-binding protein that is believed to serve as a coactivator of SRF-inducible transcription at SPES. Expression of an ATF6 ***antisense*** RNA blocked p38-mediated ANF induction through the ANF SRE. Moreover, when fused to the Gal4 DNA-binding domain, an N-terminal 273-amino acid fragment of ATF6 was sufficient to support trans-activation of Gal4/luciferase expression in response to p38 but not the other stress kinase, N-terminal Jun kinase (JNK); p38-activating cardiac growth promoters also stimulated ATF6 trans-activation. These results indicate that through ATF6, p38 can augment SRE-mediated transcription independently of Ets-related TCFs, representing a novel mechanism of SRF-dependent transcription by MAP kinases.

4/7/4 (Item 1 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

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14365027 PMID: 10358048

Phosphorylation of MAP kinases by MAP/ERK involves multiple regions of MAP kinases.

Wilsbacher J L; Goldsmith E J; Cobb M H

Department of Pharmacology, The University of Texas Southwestern Medical Center, Dallas, Texas 75235-9041, USA.

Journal of biological chemistry (UNITED STATES) Jun 11 1999, 274 (24)

p16988-94, ISSN 0021-9258 Journal Code: 2985121R

Contract/Grant Number: DK34128; DK; NIDDK

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Mitogen-activated protein (MAP) kinases are activated with great specificity by MAP/ERK kinases (MEKs). The basis for the specific activation is not understood. In this study chimeras composed of two MAP kinases, extracellular signal-regulated protein kinase 2 and p38, were assayed in vitro for phosphorylation and activation by different MEK isoforms to probe the requirements for productive interaction of MAP kinases with MEKs. Experimental results and modeling support the conclusion

that the specificity of MEK/MAP kinase phosphorylation results from multiple contacts, including surfaces in both the N- and C-terminal domains.

Record Date Created: 19990706

Record Date Completed: 19990706

4/7/5 (Item 2 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

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13022272 PMID: 8663074

A novel kinase cascade mediated by mitogen-activated protein kinase kinase 6 and MKK3.

Moriguchi T; Kuroyanagi N; Yamaguchi K; Gotoh Y; Irie K; Kano T; Shirakabe K; Muro Y; Shibuya H; Matsumoto K; Nishida E; Hagiwara M

Department of Genetics and Molecular Biology, Institute for Virus Research, Kyoto University, Sakyo-ku, Kyoto 606-01, Japan.

Journal of biological chemistry (UNITED STATES) Jun 7 1996, 271 (23) p13675-9, ISSN 0021-9258 Journal Code: 2985121R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

A cDNA encoding a novel member of the mitogen-activated protein kinase kinase (MAPKK) family, MAPKK6, was isolated and found to encode a protein of 334 amino acids, with a calculated molecular mass of 37 kDa that is 79% identical to MKK3. MAPKK6 was shown to phosphorylate and specifically activate the p38/MPK2 subgroup of the mitogen-activated protein kinase superfamily and could be demonstrated to be phosphorylated and activated in vitro by TAK1, a recently identified MAPKK kinase. MKK3 was also shown to be a good substrate for TAK1 in vitro. Furthermore, when co-expressed with TAK1 in cells in culture, both MAPKK6 and MKK3 were strongly activated. In addition, co-expression of TAK1 and p38/MPK2 in cells resulted in activation of p38/MPK2. These results indicate the existence of a novel kinase cascade consisting of TAK1, MAPKK6/MKK3, and p38/MPK2.

Record Date Created: 19960826

Record Date Completed: 19960826

